REVIEW OF LITERATURE ON HEALTH HAZARDS OF BERYLLIUM AND ITS COMPOUNDS

BY G. G. MORGIS AND J. J. FORBES

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G. G. Morgis[1/] and J. J. Forbes[2/]

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INTRODUCTION

The greatly diversified and increased uses of beryllium and its compounds in recent years have focused attention on the health hazards incident thereto. The Bureau of Mines has a fundamental interest in safety and the elimination or reduction of health hazards in the mineral and allied industries, and it recognizes that serious hazards are inherent in the production and use of beryllium and its compounds. This circular presents a review of the literature pertaining to such health hazards but does not describe original research by Bureau personnel. Most of the bibliography has been either quoted in part or abstracted in an effort to present information in a comprehensive and readable form.

In reviewing the literature, differences of opinions were recognized in the writings of the respective authors, but no attempt was made to evaluate such differences. This presentation was devoted strictly to reporting without editorial comment.

SUMMARY

Although beryllium was known to industry for about 150 years, its toxicity did not present any serious problems in the United States until the advent of World War II, when the increased use of this element and its compounds brought to light several toxicologic reactions occurring primarily in the lungs and skin of workers handling it. The pulmonary effects, characterized as acute pneumonitis and chronic pulmonary granulomatosis in beryllium workers, are far more serious than the dermatological manifestations.

The addition of beryllium to the list of toxic industrial metals has been so recent that there is still some confusion in the clinical picture. Data on magnitudes of exposure likely to be responsible for the production of disease are not yet adequate. There appears to be no relation between the severity of exposure and the development of cases - some of those having had severe exposure have not developed a reaction, whereas those having a slight exposure in some instances have developed a response.

As there seems to be no specific treatment for berylliosis outside of bed rest, oxygen, and drugs to aid respiration, environmental control of beryllium hazards should be of utmost importance. Workers who handle beryllium and members of their families who may have contact with work clothes should undergo periodic physical examinations and should be aware of the following danger signals: Skin lesions, loss of weight, fatigue, cough with or without substernal pain, shortness of breath, loss of appetite,

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general feeling of discomfort, upper respiratory infections, and increased pulse beat. The two important diagnostic aids are history of exposure and roentgenograms of the chest.

Undoubtedly, with further medical and engineering research and the continued dissemination of knowledge concerning beryllium poisoning, the fear and health hazards involved in the handling of beryllium and its compounds will diminish considerably.

HISTORICAL DISCOVERY OF BERYLLIUM

Beryllium (a grayish-white hard light metal with a steel-like luster; symbol Be; atomic weight 9.02; specific gravity 1.85; melting point 1,285°C., which is high in comparison to other light metals; boiling point 2,780°) is one of the most interesting of the minor metals and distinctly a modern development, for until the last two decades it had practically no commercial importance. Although a rare metallic element, beryllium is widely distributed in small quantities in certain feldspars, lithium, and in mica. The metal imparts many desirable characteristics to other metals when alloyed with them, thus increasing its potential use.

Beryllium stones that possess the color of light green due to traces of chromium are the well-known emerald gems. The emerald has been prized from very early times, and Cleopatra's emerald mines in upper Egypt were worked in 1650 B.C., many centuries before that famous queen saw the light.

Beryllium stones colored bluish-green by traces of ferrous iron are known as aquamarines. Queen Elizabeth of England is said to have a collection of these - her favorite stones. The aquamarine is regarded as a lucky stone.

Haüy observed that the beryl and the emerald were geometrically identical, and he asked Vauquelin (1763-1829) to compare their analyses. The beryl had hitherto been regarded as calcium aluminum silicate, but Vauquelin in 1797 showed that not only were the beryl and emerald identical chemically, but that they contained a new element, the oxide of which he called terre du Béril. This result was published in 1798, and the new earth was called la glucine at the suggestion of the editor of the Annales de Chemie et de Physique, because Vauquelin stated that its salts possess a peculiar sweet taste. The Germans, however, adopted the term Beryllerde and the name glucinum and beryllium were subsequently adopted to denote the metal itself. In 1924, the American Chemical Society decided to adopt the name beryllium instead of glucinum.

The metal itself was not isolated for many years. In 1828, Bussy and Wöhler independently obtained it by reduction of the chloride with metallic potassium. It is now usually prepared by electrolysis of the double fluoride K_2BeF_4.

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OCCURRENCE

Beryllium is never found as the native metal but occurs in nature in the form of minerals as minor constituents of certain natural ore deposits known as pegmatites. It is a bivalent element that behaves chemically much like magnesium and calcium. It occurs commonly in granitic rocks as a complex aluminum beryllium silicate known as beryl; this is the only commercial beryllium mineral. Other beryllium minerals are chrysoberyl, phenacite, and idocrase. However, beryl continues to be the primary source of beryllium. Large crystals are sometimes found and are usually uncovered in feldspar and mica mining. In such mines, a ratio of recovered beryl of 1/2 ton per 100 tons of rock moved is common. Virtually no mining operations have been started, primarily because of the presence of beryl, but this remains an important possibility.

Though beryl in museums is geneally greenish and almost always hexagonal, in the field, in its familiar pegmatites, on weathered surfaces, it is likely to assume the color of adjoining rock. The mineral beryl will scratch glass but cannot be scratched with a knife, thus distinguishing it from feldspar. The fracture does not show a distinct cleavage but generally exhibits a varying degree of transparency.

The mining of beryl deposits does not, at present, lend itself to mechanization. The supplies that one can obtain from primitive mining operations in patchy deposits of coarse-grained ore are, of course, limited and are inadequate to meet the increasing industrial demand for beryllium. As a result, the quoted price of beryl advanced from $3.00-$3.50 per short-ton unit of BeO in 1939 to $16.00-$26.00 in 1948. Although interest in mining domestic beryl was keen in 1948 and prices reached an all-time high, shipments dropped nearly 50 percent below those reported for 1947.

SOURCES AND CONSUMPTION

The fact that the potential use of beryllium exceeds the present supply indicates the immediate need for more sources of beryllium ore, particularly in this country, and the limiting of new uses for beryllium and the study of possible substitutions until such a time as more beryllium ore is available.

Domestic consumption of beryl in 1947 for the production of metal, alloys, and compounds rose to 1,735 short tons, thus exceeding by some 722 tons that for 1946 and nearly equaling the 1945 consumption of 1,738 tons. Consumers relied upon stocks of beryl transferred to industry by OMR (Office of Metals Reserve). This transfer amounted to 2,912 tons for the year.

In 1948, imported and domestically produced beryl combined were again inadequate to meet processors requirements. The shortage was alleviated by transfers to industry from the dwindling stock pile of the Office of Metals Reserve. Beryl was actively sought for the National Strategic Stock

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Pile throughout 1948. This factor probably was important in keeping prices at a high level, aside from the normal market influence of increased consumer demand. Figures on beryl stocks in the strategic stock pile are not available for publication.

The Sitting Bull and Ingersoll mines of the Black Hills Keystone Corp., both near Keystone, S. Dak., produced beryl during 1948 but were shut down in October, reportedly because of the poor demand for lithium ores. The largest production for the year from any individual property was that from the Palermo mine, near West Rumney, N. H., operated by the Ashley Mining Co. A small quantity of beryl was produced by Joe Grover from the Grover mine near Idaho Springs, Colo. Small lots reached ore buyers from many other properties of uncertain identity, principally in South Dakota, New Hampshire, Colorado, and Maine. Michael Lyons, of the Beryl Ores Co., Loveland, Colo., was active during the year as a buyer of small lots of beryl ore and held leases on a number of properties in Colorado and South Dakota. The Hyatt Berylmine, Drake, Colo., was operated in 1948 to obtain fine-grained beryliferous rock for use in the development of milling methods applicable to low-grade beryl ores. The Bureau of Mines and the Geological Survey were active during 1948 in exploring and evaluating beryl pegmatites and in developing beneficiation methods.

The Black Hills Region of South Dakota continued to be the principal domestic source of beryl, total production to date being about 1,800 tons. Important shipments have been made from Colorado (total production about 500 tons), Maine, New Hampshire, and New Mexico. Small shipments have been recorded from Connecticut, Idaho, Massachusetts, New York, North Carolina, Virginia, and Wyoming. Specimens of beryl have been found in many other States. Total reserves of inferred and indicated beryl ore in Colorado are estimated at 2,000 to 5,000 tons. Reserves of beryl at the Muscovite mine between Avon and Deary, Latah County, Idaho, have been estimated at 150 to 450 tons. The Geological Survey estimated indicated and inferred beryl reserves for Connecticut at about 860 tons.

It is disclosed that the following foreign countries produce beryl (in large, small, and/or negligible proportions) for exporting purposes as well as for their own use: Anglo-Egyptian Sudan, Argentina, Australia, Brazil, British East Africa, Canada, Finland, France, India, Kenya, Korea, Madagascar, Mexico, Nigeria, Norway, Portugal, Portuguese East Africa, Rumania, Russia, South-West Africa, Southern Rhodesia, Spain, Uganda, and Union of South Africa.

As in previous years, the producers of beryllium metal, alloys, and compounds in 1948 were the Brush Beryllium Co., Cleveland and Lorain, Ohio; Beryllium Corp., Temple, near Reading, Pa.; and Clifton Products, Inc., Painesville, Ohio. The Ceramic Division, Champion Spark Plug Co., Detroit, Mich., consumes beryl solely for dielectric manufacture. On September 21, 1948, the Brush Beryllium Co. plant at Lorain suffered a serious fire damage, which was confined to the oxide-production facilities. It was reported that Clifton Products Co. did not operate in the latter part of the year.

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USES

Beryllium metal, as such, is of far less industrial importance than its alloys with copper and eventually, perhaps, with aluminum.

The principal use so far developed for the metal is in the production of strong, heat-treatable copper-base alloys (in which the beryllium content ranges from about 0.3 to 2.5 percent) for electrical contacts, clips, small springs, and similar metallic parts. These alloys are ductile and capable of being cold-formed into various shapes, and after suitable heat treatment become very hard and have an astonishing fatigue resistance.

Extensive studies have been carried out in varous laboratories in endeavors to broaden the use of beryllium-copper alloys and to take advantage of their valuable properties of high fatigue strength, elastic limit and hardness, relatively high tensil strength and electrical conductivity, excellent corrosion resistance, good wear resistance, good resistance to galling against steel, resistance to room temperature creep (elastic drift), and various fabrication advantages associated with heat treatment or precipitation hardening. Springs made of this alloy resist fatigue and corrosion under conditons that prohibit the use of steel springs.

Were beryllium available in quantity, it would, because of its low density, be the basis for the lightest alloys one could expect to produce that would be stable in air; for example, beryllium-magnesium alloys would be exceedingly light.

At present, beryllium-copper is being used in parts of aircraft, ships, tanks, guns, wheels, instruments, engines, motors, radio, telephones, telegraph, tools, and electrical control equipment for machinery and fire protection. Where small parts are involved, design factors often explain the use of this alloy. Thus, in many aircraft instruments, beryllium-copper is used because its properties make possible the use of a smaller part.

The second most important use of beryllium, though much smaller, is in the form of oxide or other nonmetallic products - X-ray, cathode ray, radio tubes, television industry, incandescent lamps, neon-sign tubing, vacuum-tight windows for X-ray tubes, electric heating elements, phosphor compounds (luminescent indicators), ceramics, vitreous enamels, gas mantles, textile fibers, chemical and pharmaceutical products, and refractories, as, for example, certain types of crucibles. Within the last few years beryllium has assumed a new and important role in the field of atomic energy, for when mixed with radium it offers a rich source of neutrons. Certain beryllium salts having the quality, in common with several other materials, of transforming short-wave radiation and cathode rays to visible radiation are referred to as "phosphors."

Until recently, beryllium phosphors were used in about 92 percent of the production of fluorescent lamps. On May 5, 1949, Dr. Leonard A. Scheele, Surgeon General of the Public Health Service, announced that after

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June 30, 1949, beryllium phosphor no longer would be used in the manufacture of the fluorescent lamps. This was put into effect because of the possible health hazards involved in the manufacture of fluorescent lamps and in the process of disposing of burned-out bulbs containing beryllium phosphor. Cadmium is one of the substitutes for beryllium now being used as a phosphor in fluorescent lamps.

Beryllium compounds in use include beryl, beryllium carbonate, chloride, phosphate, fluoride, hydrate, lactate, nitrate, oxide, oxyfluoride, sulfate, tartrate, and zinc magnesium beryllium silicate.

OCCUPATIONAL EXPOSURE

Difference of opinion still exists as to the disease-causative properties of beryllium compounds. There is no proof as yet that beryllium is a poison in all its forms and compounds. However, cases of disease with like clinical and chemical characteristics have occurred in a variety of widely separated industries, among which the only possible common occupational exposure was to beryllium. Animal experimentation, moreover, has repeatedly demonstrated that beryllium compounds are capable of producing pulmonary diseases.

Injuries caused by beryllium compounds have not been reported as occurring in the mining (despite the general practice of hand sorting by workers), handling, and shipping of beryl, cases of disease first appearing during the industrial processes of crushing or extracting the beryllium compounds from the ore. To date, cases have been reported among workers in the processing plants, fluorescent-lamp manufacture, laboratory research, preparation of lamp phosphors, manufacture of ceramics containing beryllium, beryllium alloys, and sign tubes, working of beryllium metal, salvage of fluorescent lamps, and fabrication of crystals containing beryllium for radio use. Cases also have occurred in the neighborhood of plants as a result of exposure to exhausts containing beryllium. It is also believed that injuries may occur as a result of atmospheric contamination introduced by work clothes of an employee. In addition, cases have occurred in the atomic-energy industry, but the details of the processes involved are not generally available.

Reports from Europe of a peculiar occupational disease appeared as long ago as 1933, by Weber in Germany, Gelman in Russia, and Fabroni in Italy. Fabroni proposed the diagnostic term "berylliosis." Interest in this disease syndrome was lacking in the United States until the early years of World War II, when, as a result of the increased use of beryllium in industry, it became apparent that we faced a serious problem in occupational medicine.

In 1943, Van Ordstrand and coworkers observed men suffering from this poisoning, and in their report, published in 1945, stated that they were unable to find any reference to the toxicity of beryllium in the American literature. However, Fairhall and associates of the U.S. Public Health

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Service made an extensive study of the toxicity of beryllium and issued the National Institute of Health Bulletin 181 in 1943, entitled, "The Toxicology of Beryllium." This report indicated that beryllium metal was not considered toxic of itself; however, it was stated that the soluble compounds were irritating because of hydrolysis and should not be inhaled.

At the eighth annual meeting of the American Conference of Governmental Industrial Hygienists at Chicago, Ill., April 7-13, 1946, it was stated that although the experimental work reported in National Institute of Health Bulletin 181 indicated no toxic action of beryllium of itself, the animals used were largely guinea pigs, and that rabbits were more susceptible to the injurious effects of beryllium. The Proceedings of this eighth annual meeting further reported that:

> Consequently, further experimental work was done in which rabbits were used as experimental animals. Two groups were employed, one of which was exposed to beryllium sulfate dust and the other to potassium beryllium sulfate dust. The first group was given one exposure to beryllium sulfate, with the resulting mortality of 66 percent. The second group was given large doses of potassium beryllium sulfate dust daily for 7 days. There was no mortality in this group. This merely confirmed our earlier experiments, which indicated that the toxic action of beryllium compounds is due to the local production of hydrolytic products and not to beryllium itself. Potassium beryllium sulfate is soluble in cold water, nearly neutral in reaction, and yields all the reactions characteristic of the beryllium ion. It is not, therefore, bound as a complex which might be expected to have different properties from other beryllium-salt solutions.

Since 1943, chemical and envirmental evidence against certain beryllium compounds has been accumulating gradually. It is now generally accepted that improper contact with certain beryllium compounds may lead to serious effects on health. In view of the growing industrial application of this element, careful attention to those whose work involves beryllium exposure becomes of utmost importance.

The Quarterly Safety Summary of the Association of British Chemical Manufacturers (January-March, 1950) issued the following statement:

> A preliminary report from the Toxicology Research Unit established by the Medical Research Council, shows that soluble beryllium salts have a high degree of specific toxicity, contrary to early reports from the U.S.A. The Unit has developed a satisfactory method for the micro-determination of beryllium in body tissues, and has shown that following the intravenous injection into experimental animals of small doses of beryllium salts, a rapid focal concentration of the metal occurs within the liver which gives rise to acute liver necrosis which causes death.

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Early American work, indicating the production of malignant bone tumors in rabbits by the injection of chemically insoluble beryllium silicates, has been confirmed.

Report of the Medical Research Council, 1945-48 (CMD. 7846).

The 1949 transactions of the eleventh annual meeting of the American Conference of Governmental Industrial Hygienists lists beryllium as a substance for which no threshold limit value has as yet been established.

There is no evidence that cases of disease will occur when exposure to beryllium as oxide exceeds 100 micrograms per cubic meter of air. The disease has not occurred after 6 years of exposure to beryllium as a lamp phosphor in amounts ranging from 2 to 8 micrograms per cubic meter of air. The relationships among compounds, their solubility, particle-size distribution, type of charge, and tendency to aggregate are under investigation.

To date, in this country several hundred cases of poisoning have been attributed to exposure to the metal, usually in chemical combination, in the form of dusts, fumes, or mists. Those definitely shown to have produced various forms of beryllium poisoning are the sulfate, fluoride, oxide, chloride, oxyfluoride, carbonate, hydroxide, beryllium manganese silicate, zinc magnesium beryllium silicate, and finely powdered beryllium metal. The sulfate, fluoride, oxide, metallic dust, and perhaps the hydroxide have been shown to produce the acute disease.

Of the approximately 400 cases that have been observed or reported, by far the most have occurred in the processing of beryllium from ores; the fluorescent-lamp manufacturing industry contributed approximately 50, and the remainder were distributed in a variety of industries in different localities. Rates of attack are widely variable and usually relate to the magnitude of exposure and type of compounds.

Of special interest to physicians are two features: The extremely minute quantities of the chemical capable of causing poisoning and the long, latent period that often exists between the last exposure to beryllium compounds and the development, years later, of the form of the disease known as "delayed pulmonary granulomatosis" or "chronic pulmonary granulomatosis," which has proved to make the greatest challenge to the medical profession. The intensity of the delayed reaction and the peculiarities of the clinical picture make it unique.

Nuclear Science Abstracts for November 30, 1949, page 421, quotes Robert J. Hasterlik of the Atomic Energy Commission as stating: "It should be understood that this is a disease of the entire body although the most prominent and incapacitating features are centered in the respiratory system. Hasterlik further states:

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Tracer studies on animals with Be^7 indicate that a large and very significant portion of the injected dose of a soluble salt is rapidly deposited in the bone, where it remains almost indefinitely. After a very short period of excretion in the urine, insignificant amounts leave the animal's body. This finding of deposition in bone may give a possible clue as to the mechanism of the delayed appearance of the disease. It is conceivable that various "trigger mechanisms" act to release beryllium stored in the bones into the general circulation. It is not possible at present to set "safe" air levels. It is known that exposure to air concentrations of a beryllium compound containing 25 micrograms of beryllium per cubic meter of air has produced the acute form of the disease.

Persons of all races apparently may acquire the disease if they are exposed to beryllium. The high proportion of cases in persons of Italian, Portuguese, and French lineage, exhibited in some localities, arises from the distribution of personnel in the community and in the plant populations. Sex is not a major factor. In a few plants sufficiently large to give stable rates, the attack rates among women were in proportion to sex distribution in the plant. The first clinical evidence of chronic beryllosis has followed puerperium (a disease incident to childbirth), rheumatoid arthritis, and other diseases, each of which required hospitalization or other limitation of activity, with associated reduction in vital capacity. Age does not seem to be a factor.

Berylliosis

The two general types or variations of beryllium intoxication that are of utmost importance to all individuals who are exposed to beryllium and its compounds are dermatologic and respiratory.

Dermatologic Manifestions

Beryllium causes the same kind of damage to the skin when it gets into it as it does to the lungs when it is inhaled. The damage in both cases consists of a kind of tumor, which doctors call granuloma. Contact dermatitis from beryllium salts is like contact dermatitis from other causes. Inadequate hygienic precautions and excessive perspiration are predisposing factors.

The skin lesions associated with the industrial use of beryllium (usually being the least severe) have been somewhat overshadowed in the literature by the more serious and dramatic pulmonary lesions. However, skin lesions deserve considerable attention, for in most instances they indicate a sensitivity to the material and act as friendly sentinels.

The dermatologic reactions so far noted in those who come in contact with beryllium compounds are:

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1. Contact dermatitis. - This is usually confined to exposed parts of the body and particularly to those areas sustaining prolonged or repeated abrasion and irritation. It is often accompanied by conjunctivitis, which is an inflammation of the delicate membrane that lines the eyelids. Undoubtedly this condition is related to air concentration as well as to the working habits of the individual. Contact dermatitis tends to clear up after removal from the working environment of those affected.

The first North American clinical report on dermatology of beryllium workers was published in 1945 by Van Orstrand and coworkers. They confirmed the original European observations that dermatological manifestations consist in contact dermatitis and skin ulcer. Dermatitis occurred on the hands, arms, face, and neck of 42 of 170 workers with intense itching and often accompanied by conjunctivitis. These 42 workers showed symptoms after exposure to the dust and fumes of the sulfate, fluoride, and oxyfluoride of beryllium. Thirty eight of these cases showed acute pneumonitis.

DeNardi described this form of dermatosis as developing in 6 of 10 new workers in a plant extracting beryllium from the ore beryl. It appears to be associated only with the compounds that bear an acid radical and tends to clear up after removal from the working environment of those affected.

2. Skin ulcers. - These ulcers occur mainly among those who are exposed to beryllium sulfate and develop directly from the introduction of beryllium crystals into a minor skin cut or abrasion and occasionally just from their irritating effects when localized in a skin fold. They are raised reddish papules (pimples) with a central core in which the crystals can be found. They occur most frequently on the hands. Fortunately, a relatively efficacious method of treatment is available, by which the central core is surgically removed by curettage (scraping or cleaning).

3. Lesions arising spontaneously in patients with the chronic type of lung disease (pulmonary granulomatosis). - These skin lesions are mentioned by Hardy and Tabershaw as occurring in 2 of their series of 17 such cases. The microscopic picture was "characteristic of sarcoid" (a growth resembling a tumor).

In a paper entitled, "Beryllium Granulomatosis," Pyre and Oatway reported on a pulmonary case with skin lesions showing "typical granuloma" on biopsy (biopsy being a diagnostic examination of a piece of tissue removed from a living subject). Those patients had been in contact with beryllium phosphor for many months before skin lesions were noted, and the lesions appeared concurrently with pulmonary symptoms. In 1948, Grier and coworkers confirmed these findings in a report of two cases. They regarded the lesions as of spontaneous origin and associated with a generalized disease, as exposure to the phosphor had occurred 6 months to 5-1/2 years before onset of symptoms.

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The fact that these skin lesions develop only after the same long lag period as does pulmonary granulomatosis itself, that is, from 6 months to 5-1/2 years after cessation of work with beryllium phosphor, indicates that they are not due to external contact of the phosphor with the skin but are part of the generalized disease we now believe pulmonary granulomatosis to be.

4. Subcutaneous granuloma occurring in persons who cut themselves on fragments of broken fluorescent lamps. Grier, Nash, and Freiman, in the Journal of Industrial Hygiene and Toxicology for July 1948, present an interesting case history of a 12-year old boy referred to a hospital complaining of small, painless swellings around the angle of the jaw on the right side. His story was that 3 months previously he had been playing with some friends at a dump where there were some old fluorescent lamps. One of the boys, deciding a lamp would make an excellent baseball bat, hit a bottle with it. The lamp broke, and pieces of glass from it cut the patient on the right side of the neck. He went to his doctor, who removed some pieces of glass and cleaned and sutured the wounds. All of them except one healed perfectly well. This became a little infected and was opened a few days later. It then healed rapidly.

About 8 weeks after this, the patient thought that some small lumps were appearing beneath the scars on his face and neck. By the time 3 months had elapsed these were large enough to cause him to go to the doctor, who referred him to the hospital with a diagnosis of keloid formation (tumor made up of small fleshy masses formed in a scar). The boy's past history and family history revealed no serious disease.

On examination, some 3 months after the accident, thickened scars were present on the side of the face and neck around the angle of the jaw. Beneath each scar could be felt a firm lump, hard but not stony hard, and evidently attached to the subcutaneous tissues. The largest mass could be felt as a semi-fixed tumor about 2.0 by 2.5 cm. In all other respects the physical examination was negative.

The results of investigations were as follows: X-ray of right mandible showed a small area of rarefaction at the point of the jaw, as if a chip had been taken out of it; in the center was what appeared to be a foreign body having the density of bone. X-ray of the chest was negative. Complete blood count was normal, and blood chemical findings also were normal. The tuberculin reaction was negative.

At operation, considerable difficulty was experienced in removing the lumps because of their adherence to subcutaneous tissue. Sharp dissection was necessary, and it appeared almost as if there was some tendency for the masses to burrow into surrounding tissues. One was very firmly attached to the submandibular salivary gland. A small piece of glass was found in the largest of the lumps, but it was so small that it seemed unlikely that the lesion could be explained on the basis of an ordinary foreign-body reaction. Another small piece of glass was removed from the mandible at the point where the X-ray had shown a foreign body.

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A portion of the tissue removed was sent to Dr. Willard Machle, who analyzed spectroscopically for beryllium. It was found to contain 27 micrograms of beryllium per 100 gm. tissue. It is interesting to note that this is about the same concentration of beryllium as has been found in the lungs of those patients who have died of pulmonary granulomatosis.

Because of the danger of recurrence, treatment should involve complete excision.

The Industrial Hygiene Newsletter for July 1949 reported as follows:

> Dr. James G. Townsend, chief of the Public Health Service's division of industrial hygiene, has been serving as chairman of the Medical Advisory Committee on Beryllium to the Industry, which also includes the medical directors of the major manufacturers of fluorescent lights.
>
> In view of the fact that the manufacturing change-over in eliminating beryllium will take until the end of June 1949, and there is a stockpile of fluorescent lamps already manufactured, the Advisory Committee emphasized again the instructions it has issued in the past concerning the health hazards in the destruction of the fluorescent lights.
>
> The committee, which has been studying results of work on beryllium for three years, has reiterated its assertion to the general public that there is no danger whatever from the lights when they are intact.
>
> The possible dangers come in the destruction of old lights. Recently there have been reports of children who cut themselves on broken lights, and that the cuts healed very slowly and often suffered swelling. The committee repeated, however, its original statement that such cuts do not cause any general sickness or spread farther on the body. Surgical care is necessary if the cut refuses to heal after a period of time.
>
> Although precautions should be taken against breathing the dust from broken fluorescent lights, the committee said, there is no record of any person suffering injury from breathing of a lamp, despite the millions of lights in use.
>
> The committee reissued its instructions on the safest way to dispose of used fluorescent lights. If there are only a few lights to be broken occasionally they should be broken out of doors in a waste area or in a waste container, and the person breaking them should avoid breathing the dust or vapor that arises.
>
> The second situation in which there is a possible danger is the breaking of large numbers of fluorescent lamps, either intermittently

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or regularly, where hours may be occupied in the operation. Instructions in these cases are as follows:

(a) Break lamps out of doors in waste-disposal area or in ventilated hood. To avoid unnecessary dust, the breakage is best done within the waste container.

(b) The operator should be supplied with, and required to wear, a respirator approved by the United States Bureau of Mines for toxic dusts.

(c) Ultimate disposal of broken lamps should be such that the public and others will not be unduly exposed to powders. In situations where it is necessary to break the lamps within buildings, it should be done in an isolated room and in a hood, so as to minimize escape of dusts. Sufficient exhaust ventilation should be supplied to the hood to provide an air intake of at least 125 linear feet per minute at all hood openings.

It is recommended that broken lamps not be disposed in an incinerator, but either be thrown into water or in a dump where they are not likely to be disturbed with evolution of dust.

Significant amounts of mercury vapor may be found in the air during the breaking up of fluorescent lamps. In situations where there is frequent or continuous exposure, measurements should be made of the amounts of mercury vapor in the air during the operation. If high levels are found, protection should be provided for the operator.

Readers desiring additional and detailed information on methods and procedures for disposing of old fluorescent lamps are referred to the following:

1. Safety Reprint General No. 9, issued and distributed by National Safety Council, 425 North Michigan Avenue, Chicago 11, Ill., entitled "Safe Disposal of Fluorescent Lamp Tubes."

2. Memorandum on the precautions to be observed in breaking up fluorescent lamps, sponsored by the Medical Advisory committee on beryllium and distributed by the Saranac Laboratory, Saranac Lake, New York.

Respiratory Manifestations

The respiratory manifestations are said to be far more serious than the dermatological disclosures. The two types of respiratory disease that appear to be peculiar to industries using beryllium and that occur among workers exposed to dusts, fumes, and mists of beryllium compounds are acute pneumonitis and chronic pulmonary granulomatosis (sometimes referred to as

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chronic pneumonitis). A characteristic feature of the acute form of beryllium poisoning has been the delay between the beginning of the symptoms and the changes that take place in the lungs. In the case of the chronic disease, symptoms and lung changes often follow termination of exposure by periods of several months to 6 years. The type of reflection produced depends on many factors of exposure, including type of compound, particle size of dust or fume, extent of air contamination, duration of exposure, solubility of compound inhaled, and perhaps other factors that up to now have been overlooked.

Beryllium poisoning has not occurred from the mere handling of beryllium-copper alloy, such as is found in nonsparking tools, springs, etc. However, whenever beryllium-copper alloy is melted or machined so as to produce either a fume or a dust that contaminates the atmosphere, this disease may develop. Acute poisonings have been reported from exposure to all the soluble compounds used and from beryllium oxide, which is insoluble. There is some evidence that workers exposed to soluble salts, in whom the acute condition develops, may present the chronic form after a lapse of months to years. The evidence for this is not entirely clear, as persons showing this delayed type may have been exposed to both the soluble and the insoluble forms during their term of employment. There is no conclusive quantitative information as to the part played by dosage and particle size.

Acute pneumonitis - Acute pneumonitis of beryllium workers indicates that the reaction is largely pulmonary. Occasionally it is fatal, but many victims of it recover. Cases that do not terminate fatally (usually within 2 weeks after the onset of the disease) recover rapidly with no apparent permanent injury. The symptoms are like those of an acute chemical pneumonitis, and the course is likewise similar, though more delayed. It has been reported that an attack of acute disease has been followed by the onset of chronic berylliosis about 2 years later.

The onset tends to be insidious, beginning with soreness of nose and throat with swelling and hyperemia (a superabundance or congestion of blood in any one part) of the mucous membranes followed by a dry cough and substernal pain. The cough progresses (with variable small amounts of expectoration) for a few days to a week, when dyspnea becomes more pronounced and progressive and appears during rest. Cyanosis (a morbid condition in which the surface of the body becomes blue because of insufficient aeration of the blood) is evident. The respiratory rate is increased; anorexia (a serious nervous condition of the stomach in which the patient loses his appetite and systematically takes but little food, so that he becomes greatly emaciated) is severe, and the patient is prostrated. There is little or no fever. Principal physical findings are tachycardia (excessive rapidity in the action of the heart), scattered rales in both lungs, and decided reduction in vital capacity. Death may occur in two weeks. The course of the disease is usually 5 weeks to 4 or 5 months. All cases of acute disease observed to date have occurred during the course of employment.

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It is reported that there does not seem to be any relationship between the degree of exposure and the occurrence of the disease, as this type of beryllium poisoning can result from single excessive exposures.

Van Ordstrand stressed the importance of bed rest, possibility of recurrence on return to exposure, and desirability of change of job for recovered workers.

Chronic pulmonary granulomatosis. - This is the delayed form of the disease and the most puzzling. The onset of this disease may occur during the period of employment with beryllium or up to several years after termination of exposure to beryllium - the lag period ranges from 3 months to as much as 5 to 6 years. Physiologic strain seems to play some part in this syndrome, and undue strain apparently precipitates the disease. Although the principal manifestations are in the lungs, other organs of the body also may be affected. The chronic form is characterized by the presence of a diffuse pulmonary reaction and scattered granulomatous lesions throughout both lungs.

It has been noted that the symptomatology does not differ greatly from that of many other chronic pulmonary diseases, as the clinical manifestations resemble those of tuberculosis or almost any other type of chronic pneumonitis. In most cases this disease was first interpreted as tuberculosis until the correct diagnosis had been made. A routing examination, which is so helpful in many diseases of the chest, may be of far less diagnostic aid than history of exposure.

The first symptoms may be a mild and vague indisposition, slight but persistent loss of weight, weakness, and fatigability. There may be merely history of a cold that has persisted unusually long. In a matter of weeks or months, the symptoms that were only general, with occasional cough, gradually become more localized, and evidences of pulmonary insufficiency appear. Fatigability and weakness, which are outstanding, are usually associated with progressive loss in weight and anorexia. By and large, the cough is nonproductive, or slightly so, and worse in the morning, when after repeated bouts of coughing there is often only a small amount of expectoration, followed by some relief in the afternoon and evening. Exertion may initiate or aggravate the cough. Blood streaking is especially common in the more acute forms of the disease. The amounts of cough and sputum appear to bear no relation to the severity of disease or to the roentgenographic signs.

Machle, Beyer, and Gregorius report the following:

Because of the minimal cough in many of the chronic cases and the insidious onset (loss of weight often going unnoticed), the presenting sign frequently is exertional dyspnea.

Pain in the chest, which is prominent in the more acute forms of the disease, is sometimes momentary and overlooked by the patient with chronic disease. The pain is usually substernal and aggravated by deep breathing and coughing.

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Some increase in pulse rate occurs in the majority of cases, and an undue acceleration in pulse with activity is present in all symptomatic cases. Pulse rates are highest in the presence of cyanosis and are related to the attempts at circulatory compensation for pulmonary insufficiency.

A low or normal blood pressure is characteristic, and in several cases in which early records were obtained a reduction occurred during the course of the disease. Cyanosis and clubbing are prominent in the advanced chronic cases. Cyanosis, which is measured with difficulty, is often overlooked during the early stages of the disease but is prominent in the severe cases. Changes in the fingers usually show first as an increased curvature of the nails, followed by clubbing. Sooner or later, symptoms of cardiac decompensation (loss of power by the heart, with circulatory failure) are added to the distress of the patient. Reduction in vital capacity is in general related to the dgree of involvement of the lung by exudates, granuloma, and fibrosis. The pulmonary hypertension and strain on the right side of the heart may cause congestion of the liver, which will result in abdominal pain, nausea, and vomiting.

The roentgenologic findings may be invaluable, but they vary considerably from case to case, and a complication, such as a sudden accumulation of fluid in the pleural cavity, which is common, may change the roentgenologic picture within a few hours. As for the laboratory, it may offer interesting supplementary information; for example, there may be an increase in the number of red cells in the circulating blood, or there may be a very low oxygen tension, but the laboratory alone cannot make a specific clinical diagnosis. An electrocardiagram taken late in the disease may show signs of right ventricular strain, and repeated measurements of vital capacity may detect the progress of pulmonary damage, but the important point is this: None of these diagnostic procedures, nor all of them combined, will enable one to formulate a correct clinical diagnosis unless one is first aware that such a disease entity exists.

The essentials for diagnosis of berylliosis, as presented by Machle, Beyer, and Gregorius are:

> An occupational history of significant exposure to beryllium compounds, a characteristic type of onset and clinical disease, roentgenologic signs and clinical laboratory findings consistent with the disease, and chemical findings of beryllium in the tissues or urine. Because of the similarity of the granuloma of beryllium with that of sarcoidosis, diagnosis by biopsy is not definitive.
>
> The occupational history should be accurate and specific as to types and duration of employment, as well as to magnitude of exposure and its characteristics. The occurrence of a number of probable cases in persons who have worked in the same circumstances at the same time is strongly indicative of significant exposure.

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Diagnosis on the basis of the physical findings is not possible. The symptoms and signs, however, taken together with the characteristic onset and course plus a history of significant exposure, may lead to presumptive diagnosis.

No specific treatment has been found for the disease, though every known means has been tried to improve exchange of gases in the lungs. Pulmonary insufficiency often requires frequent or constant use of oxygen to meet needs even at rest. In the mild cases, general supportive treatment with exercise to limit of comfort is indicated; in the severer acute and chronic cases, rest in bed and continued oxygen therapy are necessary. In several cases, penicillin-streptomycin aerosol therapy was of value in dealing with intercurrent infections but did not appear to alter the course of the underlying disease.

The following two cases of chronic beryllium poisoning are cited in the Archives of Industrial Hygiene and Occupational Medicine for February 1950, pages 199-200:

CASE 1. - This is the story of a young woman who died at the age of 21. In 1941 she was employed in the manufacture of fluorescent lamps for a period of ten months. During this period she lost 5 to 6 pounds in weight. Because of this loss of weight and a general feeling of poor health, she resigned her position and procured another in an entirely unrelated industry. At this time a roentgenogram of the lungs was reported as showing slight increase in perihilar markings, with lung fields clear. For the next two years she apparently felt well, although she did not regain the weight she had originally lost. In April of 1944 she first noticed dyspnea on slight exertion. This was shortly followed by a dry cough and strangling substernal pain. She again began to lose weight, and a productive cough developed, with pleurisy and drenching night sweats. Roentgenologic examination at this time showed symmetric haziness, most marked in the bases of the lungs. She was studied at a tuberculosis sanitorium in June 1944, but no positive evidence of tuberculosis was found. She went downhill rapidly, and cyanosis developed, dyspnea increased and she had lost over 20 pounds in weight at the time of her death in December, 1944.

CASE 2. - This is the record of a young woman who died at the age of 26. From 1940 until 1944 she was employed in the fluorescent lamp industry. She spent the year 1942 in the same building as did the first patient, the one particular building in which most of the cases in this community appear to have had their origin. In 1944, while still in good health, she resigned from her position, married and moved to the West Coast. About a year later a persistent cough developed, which was variously diagnosed as a "cold," "bronchitis," "pneumonia" and lastly as "tuberculosis."

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On returning to her original community in 1946, she showed definite signs of dyspnea and loss of weight, and because of her occupational history and the roentgenologic findings her condition was diagnosed as "beryllium poisoning." She was given every form of supportive treatment, but her condition became progressively worse, and in the spring of 1948, after an attack of pneumonia, she died. During the terminal stage of her disease her vital capacity was reduced to 400 cc.

NON-OCCUPATIONAL EXPOSURE

The disease is not limited to workers but may occur in those living in the immediate vicinity of a plant or in the families of workers, who come in contact with the beryllium brought home on soiled work clothes. There have been a number of unquestionable nonworker poisonings in the families of beryllium workers. In every instance the family reported that soiled work clothes were brought home and laundered. The ultimate source of beryllium poisoning in every known instance is an industrial process.

Beginning in the latter part of 1947, a number of persons having diagnoses consistent with that of chronic pulmonary granulomatosis as seen in beryllium workers were reported among residents in the vicinity of a commercially owned plant producing beryllium compounds.

Because it was anticipated that the Atomic Energy Commission would require considerable amounts of beryllium metal and compounds in the future, it was desirable for the Commission to obtain information concerning the conditions required for the safe operation of a beryllium-producing plant. For this reason, the Commission cooperated with the Management of the beryllium-producing plant in question to determine the medical and environmental factors involved in the reported cases of berylliosis.

The results of the study are reported in the Journal of Industrial Hygiene and Toxicology for September 1949, pages 282-294, entitled "Non-Occupational Berylliosis" by Merril Eisenbud, R. C. Wanta, Cyril Dustan, L. F. Steadman, W. B. Harris, and B. S. Wolf. These authors summarized their findings as follows:

1. Eleven persons showing chronic beryllium poisoning (berylliosis) have been reported among residents in the vicinity of a beryllium-producing plant. None gave a history of occupational exposure to beryllium.

2. Ten of the affected persons resided within 3/4 mile of the plant. The distribution of the cases of berylliosis with respect to the plant indicates that the incidence of disease was a function of the concentration to which the residents were exposed.

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3. The eleventh patient resided almost 2 miles from the plant and was a member of the household of an employee of this beryllium plant. This patient's disease is believed to have resulted from atmospheric contamination introduced to the household by work clothes of the employee. Based on air analyses made during simulated home cleaning of work clothes, a daily laundering can result in the inhalation of 17 micrograms of beryllium.

4. An extensive air-analysis program conducted in the vicinity of the plant yielded useful data on the levels of atmospheric contamination. The concentration at 3/4 mile is of particular interest, because the lower concentrations beyond this distance have not produced known cases in the 7 years that this plant has been in active operation. It is estimated that the average concentration 3/4 mile from the plant during this period of time ranged from 0.01 to 0.1 microgram per cubic meter. The cases appear to have resulted from exposure prior to 1946, when the 3/4 mile concentration is estimated to have been approximately 0.1 microgram per cubic meter.

5. The average daily concentration of beryllium in the vicinity of a plant producing and processing beryllium should be limited to 0.01 microgram per cubic meter. There is no justification for applying these data as criteria in judging the hazard of exposed workers in the beryllium industry.

6. The plant in question has had a comparatively low incidence of berylliosis despite the relatively high exposure to atmospheric contamination within the plant. No satisfactory explanation can be given for this discrepancy, but the specific toxicity of the "neighborhood" contamination may be enhanced by smaller particle size than would be expected within the plant.

CONTROL OF INDUSTRIAL BERYLLIUM EXPOSURES

Both the acute and chronic forms of beryllium poisoning result from inhalation of beryllium compounds, and in several cases intoxication developed from exposures in what one would usually call nondusty atmospheres. Until such a time as engineering methods of control have been placed on a firm basis, the medical-control program should be of utmost importance.

Up to the present time it has not been possible to arrive at a threshold limit value for the various beryllium compounds with any degree of certainty. However, Stokinger and others, in their paper on Toxicity of Beryllium, report the following:

> Responses have been brought about in animals with beryllium in quantities in the order of millimicrograms. Such small quantities require a reorientation in our toxicologic thinking.

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And these amounts give rise to acute effects. It is reasonable from field survey evidence to believe that still smaller quantities produced the chronic disease in human beings and that "safe" levels of beryllium exposure may ultimately be set well below 1 microgram per cubic meter of air.

Because no safe maximum allowable concentration value has as yet been firmly established, every effort should be made to reduce to zero the dusts, fumes, and mists of beryllium compounds in air at working places. Accumulation of dusts on floors and rafters should be prohibited.

Frequent and thorough analyses of air should be a routine practice and should be mandatory. Operations that include the use of beryllium should be isolated from the general work room, and protective shields should be placed around furnaces.

High-velocity ventilation should be put into effect and, however minimal the contamination may be, all dust-producing operations should be properly ventilated. As cases are known to have occurred among residents in the vicinity of beryllium-processing plants, the effluent from exhaust systems should be filtered before they are discharged to the outside atmosphere.

Meticulous housekeeping should be maintained in all workrooms where any form of beryllium might be handled. Dry sweeping should be avoided, and cleaning should be done by wet methods or after working hours with a vacuum cleaner by a worker provided with a respirator. Spills should be cleaned up promptly, and no beryllium-containing materials should be allowed to remain on the floors where they could be ground into dust underfoot.

Protective clothing, gloves, and masks should be used even with high-velocity ventilation. The masks should be of a type approved by the Federal Bureau of Mines for use when dealing with toxic materials.

No food should be eaten or stored in any workroom where beryllium is handled, nor should smoking be permitted.

Care should be taken in the disposal of fluorescent lamps to avoid inhalation of the powder or cuts from the glass when the tubing is broken.

Careful attention should be given to all breaks in the skin of workers handling beryllium, particularly phosphor-coated glass, to insure that particles of the compound are not retained subcutaneously. Workers should be instructed to report promptly any minor skin injury, symptom, or lesion to the plant medical office. Dermatitis and skin ulcers may be prevented by preemployment instructions as to hazards, use of protective devices and clothing, and personal hygiene.

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Periodic medical examinations, including a weight check and a chest X-ray, are advisable for all workers exposed to beryllium metal, alloy, oxide, or other compounds in the form of dusts, fumes, or mists. Workers should practice constant personal vigilance.

Continued cooperation by physicians, research workers, and industrial hygienists is necessary to insure complete control of beryllium hazards in industry.

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